describe how in the hyphae of C. *albicans*, the *cpp1/cpp1* mutation activates transcription of a putative virulence determinant, the secreted aspartyl proteinases SAP4–6. In an *efg1/efg1* mutant, SAP4–6 gene expression is completely absent. Hence, *cek1/cek1* reverted the morphologic phenotypes of *cpp1/cpp1*. The *cpp1/cpp1 cek1/cek1* double mutant maintains overexpression of SAP4–6. We detected differences in putative Ste12p and Tec1p transcription factor binding sites in the 5-UTRs of the SAP4–6 isogenes. A functional significance for these features was established by ribonuclease protection assay-derived measurements of transcriptional activity at the SAP4–6 isogene loci. A putative *C. albicans* TEC1 homolog was identified, which encodes a TEA/ATTS DNA-binding domain containing polypeptide

O53 Differential expression of secreted aspartyl proteinases by RT-PCR and immunoelectron microscopy in a model of human oral candidosis and in patient samples from the oral cavity

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Objectives: To study the involvement of secretory aspartyl proteinases (Sap), a possible virulence factor secreted by *Candida albicans*, in an in vitro model of oral candidosis and in vivo

Methods: We examined the temporal regulation of the mRNA expression of seven known members of the SAP gene family by RT-PCR in (1) an in vitro model of oral candidosis based on reconstituted human epithelium (RHE) and (2) in clinical samples from patients with oral candidosis

Results: SAP1 and SAP3 transcripts were first detected 42 h after inoculation of RHE, while at the same time slight morphologic alterations of the epithelium were documented by light microscopy. SAP6 expression occurred 6 h later concomitantly with germ tube formation of some infecting *Candida* cells and severe lesions of the epithelial tissue. SAP2 and SAP8 RT-PCR products were first detected 60 h after infection, while SAP4 and SAP5 transcripts were never discovered. Thus, a temporal progression of SAP expression in the order SAP1 and SAP3, SAP6, SAP2 and SAP8 was observed at the same time as increasing RHE damage occurred. Expression of SAP1, SAP2, SAP3 and SAP6 was also detected by RT-PCR in samples from patients suffering from oral candidosis

Conclusions: Our results suggest that the pathogenesis of experimental and clinical oral candidosis is associated with differential and temporal regulation of SAP gene expression

O54 Attenuated virulence of different SAP (secretory aspartyl proteinase) null mutants in an in vitro model of human oral candidosis

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Objectives: To examine the precise role of Saps in the development of histologic alterations in a model of human oral candidosis

Methods: On the one hand, we studied the effect of the aspartyl proteinase inhibitor pepstatin A on the experimental infection of reconstructed human epidermis in vitro. On the other hand, the virulence of different SAP null mutants (delta sap1; delta sap2; delta sap3; delta sap1,3 and delta sap4–6) in relation to yeast cells of the wild-type strain of *Candida albicans* was tested. The SAP gene expression of the wild-type strain and the mutants was investigated by RT-PCR

Results: The histologic lesion caused by the wild-type strain was strongly reduced by pepstatin A and to a lower extent with respect to infection with the delta sap1,3 mutant. A somewhat smaller but equal reduction of the histologic damage was observed for each of the single SAP null mutants. The histologic lesion due to the delta sap4–6 mutant was increased in comparison to the wild-type strain. In comparison with the SAP gene expression pattern of the wild-type strain, an earlier onset of SAP8 expression was exclusively demonstrated by the delta sap1 and the delta sap1,3 mutants

Discussion: The results suggest that SAP1–3 but not SAP4–6 may be important for this type of candidosis, both in vitro and in vivo

Inflammatory host responses

O55 The biological role of lipopolysaccharide (LPS) binding protein (LBP) during acute phase, sepsis and SIRS

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Objectives: During sepsis and SIRS, the host organism mounts the acute-phase response, including the upregulation of several hepatic acute-phase reactants (APRs). LPS binding protein (LBP) is an APR that binds LPS and phospholipids. In vitro, LBP in low concentrations has been shown to transfer LPS to its cellular receptor CD14, enabling cellular responses such as cytokine production. Here we investigate the role of constitutive and acute-phase concentrations of LBP in vitro and in vivo in order to elucidate host defense mechanisms during sepsis and SIRS

Methods: Recombinant murine LBP was expressed in a baculovirus system. A murine sepsis model was established, and the $LBP^{-/-}$ mouse was employed. The murine macrophage cell line RAW 264.7 was instrumental for assessing LBP activity in vitro. Additionally, sera from sepsis and SIRS patients were analyzed for LBP content by ELISA. The biological activity of these sera was assessed in different assays for their LPS-transfer and TNF-a-inducing capacity employing a FACS assay and FITC-labeled LPS, and a monocyte stimulation assay involving TNF-a-ELISA

Results: High concentrations of LBP in vitro suppressed LPS-mediated TNF-a and in a murine peritonitis model suppressed cytokine production, hepatic failure and lethality. Although more resistant against LPS, the $LBP^{-/-}$ mouse is more susceptible to a bacterial infection. Sera from sepsis and SIRS patients containing high concentrations of LBP failed to enhance LPS transfer to monocytes and blocked LPS-induced TNF-a synthesis

Conclusions: LBP in vivo may have a dual function regarding the mediation of LPS effects towards host cells. While constitutive levels enhance LPS effects, acute-phase levels diminish LPS activity and thus may protect the host

O56 Endotoxin neutralization by LBP199–IgG1 Fc chimeric molecules

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Objectives: The use of agents that neutralize lipopolysaccharide (LPS) has been proposed as adjunctive therapy for treatment of serious Gram-negative bacterial infections. Monoclonal anti-LPS antibodies (mAbs) were not able to neutralize LPS derived from a broad array of Gram-negative organisms. LPS-binding protein (LBP) binds with high affinity to lipid A derived from a variety of Gram-negative bacteria. It could be demonstrated that the LPS-binding domain of LBP is localized in the first N-terminal 199 amino acids. This fragment was shown to inhibit endotoxin function

Hypothesis: A fusion protein (FP) that combines the LPS-binding motif of LBP with the constant domain (Fc part) of immunoglobulin G (IgG) will exhibit LPS neutralization and Fc receptor-mediated cell binding

Methods: $LBP_{(199)}$ cDNA was obtained by RT-PCR and cloned in front of the Fc part of IgG_1. After transient overexpression in CHO cells, LBP_{199}–IgG_1 Fc chimeras were detected in the culture supernatants by Western blotting

Results: Transfected CHO cells secreted LBP_{199}–IgG_1 Fc FP, which was purified. A control polyacrylamide gel shows a 56-kDa band corresponding to the predicted size of the LBP_{199}–IgG_1 Fc FP. Purified LBP_{199}–IgG_1 Fc FP specifically binds to *Escherichia* coli LPS compared to IgG_1 Fc ($p<0.02$). Human monocytes treated with LBP_{199}–IgG_1 Fc FP secreted significantly less TNF-a in response to 1 ng LPS than cells treated with the purified IgG_1 Fc or untreated cells (233.9 pg/mL versus 470.6 pg/mL versus 917.3 pg/mL respectively, $p<0.02$)

Conclusions: These data demonstrate that LBP_{199}–IgG_1 Fc chimeras are able to exhibit biological activity in vitro. In principle, these agents will bind to and neutralize LPS from different Gram-negative bacteria (LBP_{199} properties), and fix complement and bind to phagocytotic receptors (IgG Fc properties). Such FPs may be extremely useful in the treatment of severe Gram-negative bacterial infections

O57 Murine lipoproteins inhibit TNF-α release by macrophages upon LPS challenge in the presence and absence of LPS binding protein (LBP)

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Objectives: Bacterial lipopolysaccharide (LPS) stimulates monocytes and macrophages for release of pro-inflammatory cytokines like TNF-a, IL-1 and IL-6, potentially leading to sepsis. Serum has been shown to lessen LPS-mediated effects towards macrophages in vitro. We have recently shown that LPS binding protein in high concentrations (hdLBP) can also inhibit LPS effects in a mouse macrophage cell line in vitro and in a murine sepsis model (Lamping et al, J Clin Invest 1998;101: 2065). In this study we analyze the inhibitory activity of serum compounds towards mouse macrophages stimulated by LPS and LBP

Methods: Mouse serum was collected from wild-type and $LBP^{-/-}$ mice. Lipoproteins and lipoprotein-deficient sera were prepared by sequential flotation ultracentrifugation. The murine macrophage cell line RAW 264.7 was stimulated with *E. coli* 0111:B4 LPS and recombinant murine LBP in the absence and presence of varying concentrations of sera and lipoproteins. Supernatants of RAW 264.7 cells were assessed for TNF-a content by ELISA

Results: Inhibition of LPS-induced TNR-a release was similar in LBP-knockout and wild-type murine serum. In contrast, lipoprotein-deficient serum failed to block LPS effects at low concentrations. Additionally, lipoproteins, in the absence and presence of LBP, blocked TNF-a release induced by LPS

Conclusions: hdLBP and lipoproteins are both inhibitors of LPS-induced TNF-a release. Furthermore, the inhibitory activity of both compounds appears to be independent in serum, with lipoproteins being the major factor for LPS inhibitory activity

O58 Bacterial cell wall components activate brain microvascular endothelial cells involving activation of MAP kinases, resulting in TNF-a and NO release

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Objectives: Recently, *Streptococcus pneumoniae* has become the most common cause of bacterial meningitis in adults. Cell wall components from *S. pneumoniae* (PCW) have been shown to induce meningeal inflammation and activate cerebrovascular endothelial cells (BMECs). These cells constitute the blood–brain barrier, whose breakdown is a crucial event during bacterial meningitis. Here we investigate the molecular mechanism involved in this inflammatory process

Methods: Tyrosine phosphorylation of mitogen-activated protein (MAP) kinases was assessed by Western blotting, and the kinase activity in lysates of cultured BMECs was detected by radioactive assays. TNF-a and NO release was determined by ELISA

Results: Recently we demonstrated that BMECs release TNF-a and NO upon stimulation with both LPS and PCW. Here we investigate the involvement of MAP kinases in the signaling cascade leading to TNF-α and NO release. LPS and PCW induced the tyrosine phosphorylation and activation of MAPK erk-1 and erk-2, and p38. Surprisingly, both LPS and PCW induced MAPK activation in an LPS binding protein (LBP)-dependent fashion. Furthermore, genistein, a tyrosine kinase inhibitor, reduced LPS- and PCW-induced TNF-a and NO release to 21% and 66% respectively, whereas SB 203580, a specific inhibitor of the p38 MAPK, had no effect

Conclusion: These studies provide evidence that in addition to LPS activation, PCWs stimulate brain endothelial cells for an inflammatory response involving p42/p44 MAPK erk-1 and erk-2

O59 Blood-brain barrier disruption by *Streptococcus pneumoniae* in an in vitro model of pneumococcal meningitis

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Objectives: To investigate the influence of *Streptococcus pneumoniae* on the blood–brain barrier (BBB) integrity during meningitis

Method: An in vitro model of the BBB consisting of primary cerebral endothelial cells in coculture with astrocytes has been used. To determine the tightness of the endothelial cell monolayer, the transendothelial electrical resistance (TEER) was measured over 24 h. A type 1 pneumococcal strain was used for the stimulation experi-

ments in the absence and presence of anti-TNF-a antibodies, a PAF receptor antagonist, and cycloheximide

Results: The TEER decreased rapidly after adding pneumococci into the endothelial compartment (A) or the astrocyte-containing compartment (B). (A) The breakdown of TEER was not influenced by the PAF receptor antagonist WEB 2187 and was independent of TNF-a. (B) In the presence of TNF-a neutralizing antibodies, the TEER recovered after 6–8 h. Cycloheximide abolished the effect of pneumococci if bacteria were added into the endothelial or into the astrocyte-containing compartment

Conclusion: Pneumococci decreased endothelial tightness independently of an interaction with the PAF receptor by an up to now unknown pathway. Once the bacteria get into contact with astrocytes, TNF-a is released, potentiating the loss of BBB tightness. The decrease of TEER was dependent on de novo protein synthesis in the used in vitro model

O60 Rifampicin reduces mortality in experimental *Streptococcus pneumoniae* meningitis

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Objective: Rifampicin releases smaller quantities of pro-inflammatory cell wall products from *Streptococcus pneumoniae* than β-lactam antibiotics in vitro and is capable of activating the glucocorticoid receptor. Since the host's inflammatory response contributes to mortality and neurologic sequelae in meningitis, we studied rifampicin versus ceftriaxone in a mouse model of *S. pneumoniae* meningitis

Methods: C57B1 mice infected intracerebrally with *S. pneumoniae* were treated either with the non-bacteriolytic rifampicin or with the β-lactam ceftriaxone (n=43 each) 2 mg subcutaneously every 12 h for 3 days. After an observation period of another 3 days, mice were killed, and hematoxylin–eosin stains of coronal brain sections from these animals were performed to assess neuronal damage

Results: Rifampicin reduced overall mortality from 49% to 26% (p=0.04). Kaplan–Meyer analysis revealed a substantial reduction of mortality during the first 24 h in mice receiving rifampicin (difference in survival time: p=0.007). In mice treated with rifampicin, 8 h after a subcutaneous dose of 21 mg, concentrations of lipoteichoic and teichoic acids were lower in serum (medians: <0.5 versus 27.0 ng/ml, p=0.02) and cerebrospinal fluid (pooled specimens: 97.5 versus 206.0 ng/ml) than in mice treated with ceftriaxone. Mice dying in the acute phase had severe brain edema. No substantial differences in neuronal damage were observed in rifampin- and ceftriaxone-treated mice surviving the acute phase of meningitis

Conclusion: The use of rifampicin reduces the release of pro-inflammatory cell wall components from *S. pneumoniae* and decreases overall mortality in experimental pneumococcal meningitis by a strong decline of the mortality within the first 24 h

O61 *Chlamydia pneumoniae*-mediated endothelial cell activation

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Objectives: *Chlamydia pneumoniae* is an important respiratory pathogen. Recently, its presence has been demonstrated in atherosclerotic lesions. In this study we characterized the effects of *C. pneumoniae* on endothelial cell activation with impact on the expression of endothelial adhesion molecules followed by subsequent monocyte adhesion and transmigration.

Methods: Human umbilical vein endothelial cells (HUVECs), human aortic endothelial cells (HAECs), counterflow centrifugation, cell surface ELISA, parallel plate flow chamber, transmigration assay, EMSA, reporter-gene assay, Northern blot, Western blot.

Results: Infection of HUVECs and HAECs with *C. pneumoniae* dose- and time-dependently increased endothelial expression of E-selectin (4 h), intercellular adhesion molecule-1 (ICAM-1, 12 h) and vascular cell adhesion molecule-1 (VCAM-1, 24 h) with subsequently enhanced monocyte adhesion as well as transmigration. These effects were blocked by monoclonal antibodies against endothelial and/or leukocyte adhesion molecules (b_1- and b_2-integrins). Additionally, activation of two different signal transduction pathways in *C. pneumoniae*-infected endothelial cells was shown: a rapid increase in total protein tyrosine phosphorylation with upregulation of phosphorylated p42/p44 MAPK, and NF-kB activation/translocation with subsequent enhanced mRNA expression for the adhesion molecules E-selectin, ICAM-1 and VCAM-1.

Conclusion: *C. pneumoniae* triggers a cascade of events that could lead to endothelial activation, inflammation and thrombosis, which in turn may result in or may promote atherosclerosis

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O62 Cytoplasmic fragmented DNA colocalizes with *Chlamydia pneumoniae* antigens in atherosclerotic lesions

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Objectives: To find support for the theory that antigens of *C. pneumoniae* persist in atherosclerotic lesions, while nucleic acids are degraded

Methods: Specimens from in vitro infected cells were obtained 3 days after infection. Human specimens of carotid arteries were obtained at autopsy. Sections were stained by immunocytochemistry (ICC) using monoclonal antibodies. Adjacent sections were stained after in situ hybridization (ISH) for detection of DNA or 16S rRNA and were further analyzed by the in situ DNA end labeling (TUNEL) assay to detect fragmented DNA

Results: The development of inclusions in in vitro infected cells was monitored by all techniques. All ICC and ISH techniques were clearly positive 29 h after infection. Staining of both ICC and ISH showed compact inclusions. The TUNEL assay was negative on these sections, but showed uniformly sized dots in the cytoplasm of infected cells after treatment with DNase. The antigen-staining pattern of human specimens was different: granular, dispersed throughout the cytoplasm. ISH for detection of DNA or 16S rRNA was negative. Uniformly sized dots stained by the TUNEL assay were found in the cytoplasm in five human specimens, of which four were

also positive for *C. pneumoniae* antigens. Cells positive for *C. pneumoniae* colocalized in adjacent sections with TUNEL-positive cells, suggesting that the fragmented DNA could originate from *C. pneumoniae*

Conclusions: *C. pneumoniae* 16S rRNA and DNA are not detectable in advanced atherosclerotic lesions. *C. pneumoniae* DNA might be heavily degraded. *C. pneumoniae* antigens persist in macrophages, but not viable bacteria.

O63 *Chlamydia pneumoniae* infection induces the development of antibodies to HSP60 in rabbits

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Objectives: *Chlamydia pneumoniae* infection is associated with arteriosclerosis. Intranasally inoculated *C. pneumoniae* has been shown to induce inflammatory arteriosclerosis-like changes in the aortas of rabbits by 5 weeks after infection and its presence in produced lesions has been demonstrated. Several reports have demonstrated the involvement of *C. trachomatis* HSP60 proteins in tissue damage after repeated infections. Our purpose was to measure *C. pneumoniae* HSP60 antibodies in the sera collected from rabbits infected intranasally with *C. pneumoniae* to see whether these antibodies are formed in response to infection and if their presence correlates with the inflammatory findings in rabbit aortas

Methods: The sera of 22 New Zealand White rabbits infected twice intranasally with *C. pneumoniae* were tested by enzyme immunoassay for the presence of *C. pneumoniae* HSP60 and *Mycobacterium bovis* HSP65 antibodies

Results: HSP60 antibodies increased as a function of time up to the fifth week from infection. Samples from uninfected animals did not contain antibodies against the HSP60 proteins. All infected animals had high antibody titers at the end of the experiment, when six of nine rabbits had developed arteriosclerotic changes. There was a significant correlation (0.79) between *C. pneumoniae* HSP60 and *M. bovis* HSP65 antibody titers. Thus, intranasal infection with *C. pneumoniae* had led to the development of cross-reactive *M. bovis* HSP65 antibodies in addition to specific antibodies

Conclusions: *C. pneumoniae* HSP60 antibodies were formed in rabbits infected with *C. pneumoniae* simultaneously with the development of arteriosclerotic changes in aortas, and these animals showed cross-reactive responses to HSP60 protein, suggesting the possibility of an autoimmune reaction as well. Further studies are needed to resolve the role of HSP60 proteins in the pathogenesis of arteriosclerosis

O64 Assessment of regional cerebral blood flow in patients with bacterial meningitis by dynamic computed tomography

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The aim of the study was to determine correlations between changes in cerebral blood flow in patients with bacterial meningitis and neurologic symptoms, severity of the disease assessed on the basis of the Glasgow Coma Scale (GCS), pleocytosis and protein concentration in cerebrospinal fluid (CSF). Dynamic CT, which consists of doing rapid scans of the same layer after administration of iodic contrast, was done in 34 patients with bacterial meningitis. The regional blood supplied was assessed by measurement of medium value of radiation attenuation coefficient in the white matter of frontal and occipital horns of lateral ventricles, symmetrically in both hemispheres. Dynamic CT was done 3–4 days after the onset of symptoms and was repeated after 6–8 weeks. Differentiation in the regional blood flow during the acute phase of the disease was observed. Reduction of the regional perfusion in the acute phase compared to the convalescent phase of the disease was demonstrated. In both the acute and convalescent phases, perfusion was higher in the posterior part of the brain. In some patients, a relationship between the appearance of neurologic symptoms (seizures, pyramidal syndrome, cranial and peripheral nerve palsies) and changes in blood flow in particular regions of the brain was noticed. In patients who aggregated low GCS scores, inflow of the blood was high, and in patients who were in better condition, inflow was smaller. High pleocytosis in the CSF was associated with small blood inflow and perfusion, whereas high protein concentration correlated with higher inflow and increase in regional perfusion

Molecular diagnostic methods

O65 Performance of the Becton Dickinson BDProbeTec ET System in detecting *Chlamydia trachomatis* and *Neisseria gonorrhoeae* from endocervical and urine specimens in women

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Objective: To evaluate the performance of the BDProbeTec ET System for the detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* in women having a low prevalence of infection

Method: Endocervical and urine specimens were obtained from 507 female patients attending family planning clinics. Each specimen was tested using the BDProbeTec ET System, conventional culture techniques, and LCR technology for the presence of *C. trachomatis* and *N. gonorrhoeae*. BDProbeTec ET System performance was compared to an 'enhanced' gold standard. The 'enhanced' gold standard is defined as a laboratory diagnosis of infection based upon the combined criteria of culture, another nucleic acid amplification test (LCR), and direct fluorescent antibody

Results: The sensitivity of the BDProbeTec ET System *C. trachomatis* assay for endocervical swab specimens was 95.5%, with a specificity of 99.1%, and for urine specimens the sensitivity was 100%, with a specificity of 98.4%. The sensitivity of the BDProbeTec ET System *N. gonorrhoeae* assay for endocervical swab specimens was 100%, with a specificity of 99.6%, and for urine specimens the sensitivity was 80.0%, with a specificity of 99.2%

Conclusion: The BDProbeTec ET System is a highly sensitive and specific DNA amplification assay for the detection of chlamydial and gonorrheal infections in women for both endocervical and urine specimens.

O66 Comparative evaluation of three commercial quantitative HBV DNA assays

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Objectives: In the present study we comparatively evaluated three commercially available assays for quantification of hepatitis B virus